Cognitive function, self-awareness, and neuroimaging findings in obsessive-compulsive presentations after traumatic brain injury

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COGNITIVE FUNCTION, SELF-AWARENESS, AND NEUROIMAGING FINDINGS IN OBSESSIVE-COMPULSIVE PRESENTATIONS AFTER TRAUMATIC BRAIN INJURY.

Funcionamiento cognitivo, auto-conciencia y hallazgos en neuroimágenes en síntomas obsesivo-compulsivos posterior a una lesión cerebral traumática

Achados do funcionamento cognitivo, autoconsciência e neuroimagem em sintomas obsessivo-compulsivos após lesão cerebral traumática

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Key words: Traumatic brain injury; Obsessive-compulsive disorder; Obsessive-compulsive personality disorder; Self-awareness; Cognitive impairment

Palabras clave: Lesión cerebral traumática; Desorden obsesivo compulsivo; Trastorno de personalidad obsesivo compulsivo; Autoconsciencia; Deterioro cognitivo

Palavras-Chave: Traumatismo cranioencefálico; Transtorno obsessivo-compulsivo; Transtorno obsessivo-compulsivo da personalidade; Autoconsciência; Comprometimento cognitivo

A B S T R A CT:

This prospective study examined associations between cognitive functions, self-awareness, neuroimaging data, and obsessive-compulsive disorder symptomatology in a sample of 31 patients with moderate-severe Traumatic Brain Injury (TBI). *Methods.* Participants completed neuropsychological tests examining specific aspects of executive functioning, as well as new learning and retention. Questionnaires assessing obsessive-compulsive disorder (OCD) symptoms and obsessive-compulsive personality disorder (OCPD) traits were completed. Patients and their treating clinicians independently completed the same questionnaire to determine level of self-awareness (SA). Discrepancy scores were used as a measure of SA. *Results.* Standard frequentist statistics were calculated, supplemented with Bayesian analysis. Bayes factors showed strong support for the presence of a correlation between Rey Complex Figure (RCFT) strategy and the Florida Obsessive-Compulsive Inventory (FOCI) symptom scores, and moderate support for the presence of a correlation between RCFT strategy and FOCI severity scores. Conclusion. Overall, results indicate self-report of new onset obsessions and compulsions after TBI were associated with specific executive functions rather than memory and retention. This study suggests that OCD phenomena after TBI may in part be explained by the presence of specific cognitive deficits. Accurate differential diagnosis of OCD, versus cognitive impairment masquerading as OCD after TBI, has implications for the treatment and rehabilitation of patients.

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R E S U M E N:

Este estudio prospectivo examinó la asociación entre funciones cognitivas, autoconciencia, datos de neuroimagen y la sintomatología obsesivo-compulsiva en una muestra de 31 pacientes con lesión cerebral traumática (LCT) moderada a grave. *Método:* Se aplicaron tanto pruebas neuropsicológicas para examinar aspectos específicos del funcionamiento ejecutivo como para medir nuevo aprendizaje y retención. Se completaron cuestionarios que evalúan los síntomas del trastorno obsesivo compulsivo (TOC) así como los rasgos del trastorno obsesivo compulsivo de la personalidad (OCPD). Los pacientes como sus médicos tratantes completaron de forma independiente el mismo cuestionario para determinar nivel de autoconciencia (SA). *Resultados:* Se calcularon estadísticas frecuentistas estándar, complementadas con análisis bayesiano. Los factores de Bayesianos mostraron un fuerte apoyo a la presencia de una correlación entre la estrategia de Rey Complex Figure (RCFT) y los puntajes de síntomas del Inventario Obsesivo-Compulsivo de Florida (FOCI), y un apoyo moderado a la presencia de una correlación entre la estrategia RCFT y los puntajes de severidad de FOCI. *Conclusión.* En general, los resultados indican que el autoinforme de nuevas obsesiones y compulsiones después de que las LCT se asociaron con funciones ejecutivas específicas en lugar de memoria y retención. Este estudio sugiere que los fenómenos de TOC después de una LCT en parte pueden explicarse por la presencia de déficits cognitivos específicos. El diagnóstico diferencial preciso del TOC frente al deterioro cognitivo disfrazado de TOC después de una LCT tiene implicaciones para el tratamiento y la rehabilitación de los pacientes.

R E S U M O:

Este estudo prospectivo examinou a associação entre funções cognitivas, autoconsciência, dados de neuroimagem e sintomatologia obsessiva compulsiva em uma amostra de 31 pacientes com lesão cerebral traumática (TCE) moderada a grave. *Método:* Testes neuropsicológicos foram aplicados para examinar aspectos específicos do funcionamento executivo e medir novas aprendizagens e retenções. Questionários que avaliam os sintomas do transtorno obsessivo-compulsivo (TOC), bem como os traços do transtorno obsessivo-compulsivo da personalidade (TOCD), foram concluídos. Os pacientes, bem como os médicos em acompanhamento, preencheram independentemente o mesmo questionário para determinar o nível de autoconsciência (SA). *Resultados:* As estatísticas de frequência padrão foram calculadas, complementadas pela análise bayesiana. Os fatores bayesianos mostraram forte apoio à presença de uma correlação entre a estratégia Rey Complex Figure (RCFT) e os escores de sintomas do Florida Obsessive-Compulsive Inventory (FOCI), e apoio moderado à presença de uma correlação entre a estratégia RCFT e os escores de gravidade do FOCI. *Conclusão* Em geral, os resultados indicam que o autorrelato de novas obsessões e compulsões após TCEs foi associado a funções executivas específicas, em vez de memória e retenção. Este estudo sugere que os fenômenos do TOC após o TCE podem ser parcialmente explicados pela presença de déficits cognitivos específicos. O diagnóstico diferencial preciso de TOC versus comprometimento cognitivo disfarçado de TOC após o TCE tem implicações no tratamento e reabilitação dos pacientes".

Introduction

While it is well-established that moderate-severe Traumatic Brain Injury (TBI) almost invariably results in a combination of cognitive impairment, physical problems, and behavioural changes, a broad range of neuropsychiatric consequences such as anxiety and depression are relatively common also. These neuropsychiatric co-morbidities can increase the complexity of diagnostic formulation and the rehabilitation these patients may sometimes require. While some research has been undertaken on obsessive-compulsive symptoms after TBI, the literature remains fairly limited. Although not the most frequently encountered neuropsychiatric disorder after TBI, Obsessive-Compulsive Disorder (OCD) symptoms have nevertheless been reported after TBI (Gould, Ponsford &Spitz, 2014; Hofer, Frigerio, Frischknecht, Gassmann, Gutbrod, 2013; Coetzer, 2004 & 2011; Grados, 2003; Williams, Evans, & Fleminger, 2003; Berthier, Kulisevsky, Gironell &López, 2000; Childers, Holland, Ryan & Rupright, 1998; Berthier, Kulisevsky, Gironell & Heras, 1996; Kant, Smith-Seemiller & Duffy, 1996; Mallya, Sutherland, Pongracic, Mainland & Ornstein, 2015). The prevalence of OCD in TBI patients varies, with up to 14% of individuals with TBI reporting new-onset OCD (Berthier, Kulisevsky, Gironell &López, 2000). An important clinical and research question is to determine whether OCD secondary to TBI is a likely consequence of psychological, environmental, or biological factors, or indeed a combination of these.

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Research findings from different studies have directly and indirectly provided potentially converging findings on how OCD after TBI might occur. These can broadly be divided into neuroimaging studies in patients with either idiopathic OCD, or post-TBI OCD, and studies of cognitive function in both populations also. Neuroanatomical models of OCD in patients without TBI generally seem to suggest the presence of fronto-striatal pathophysiology (Melloni, Urbistondo, Sedeño, Gelormini, Kichic & Ibanez, 2012; Rasmussen, Eisen & Greenberg, 2013). Interestingly, some studies have linked damage to frontal, temporal, and cingulate brain regions, and the basal ganglia, or both, in patients with OCD secondary to TBI (Coetzer, 2011; Grados, 2003). Furthermore, Figee, Wielaard, Mazaheri and Denys (2013) looked at data from 37 patients with brain injury, and concluded that damage to the cortico-striato-thalamic circuit, temporal and parietal cortex, the cerebellum and brainstem were associated with obsessive behaviours. But what about cognitive functions in patients with OCD who do not have a history of TBI? The neuropsychology of idiopathic OCD has been extensively researched, but conflicting results have been reported as regards the presence of cognitive impairment. Nevertheless, generally impairments of various executive, processing speed, visual-spatial, and memory functions have been identified as possible contributors to OCD in this population (Abramovitch, Abramowitz & Mittelman, 2013).

On the other hand, cognitive impairments including in the areas of memory, executive functioning, attention, and processing speed are frequently observed in those patients who present with OCD following TBI (Gould, Ponsford &Spitz, 2014; Coetzer, 2004; Berthier, Kulisevsky, Gironell & Heras, 1996). However some, if not most, of these cognitive impairments are also commonly present in many patients with TBI who do not have co-morbid OCD or indeed another psychiatric diagnosis, including for example anxiety disorders. This raises the question of whether there is an association between a specific pattern of cognitive impairment secondary to TBI and an increased likelihood of these patients then presenting with post-TBI OCD phenomena. Naturally it should also be kept in mind that the presence of a psychiatric disorder such as OCD where anxiety is a part of the clinical presentation, can potentially also be because of anxiety adversely affecting a patients' cognitive functioning after TBI.

The above brief overview of the current literature suggests that the exact biological and cognitive basis for OCD post-TBI still remains relatively poorly understood. More recently Rydon-Grange and Coetzer (2015) provided an overview of the literature on OCD phenomena after TBI and concluded that further research was needed to disentangle the complex interaction between cognitive impairment and obsessive-compulsive spectrum presentations such as OCD and Obsessive-Compulsive Personality Disorder (OCPD) secondary to TBI. Cognitive impairments associated with TBI may mimic some features of idiopathic OCD. For example, memory deficits following TBI may result in excessive checking of tasks, mirroring compulsions. Likewise, executive function impairments resulting in perseverative behaviour may mirror the repetitive behavioural patterns typical of OCD, and reduced processing speed or even poor set-shifting following TBI may resemble obsessional slowness. The current study is an extension of previous work by Rydon-Grange and Coetzer (2017), to examine the potential association between specific aspects of executive function including strategy, speed-accuracy trade off (i.e. prioritising copying a line drawing to near perfection, but taking much longer than would normally be the case), memory, and self-awareness in OCD presentations after TBI.

Understanding more about possible cognitive factors contributing to OCD-type presentations after TBI is important from a rehabilitation perspective. In particular, determining whether particular symptoms may primarily reflect the well-known cognitive impairments characteristic of TBI, or alternatively, new-onset OCD, is of clinical relevance for the rehabilitation of these patients. 'OCD' associated with TBI, if primarily a function of cognitive impairment, may respond better to cognitive rehabilitation, whereas 'true' OCD after TBI, may require an altogether different treatment approach, including pharmacological and psychological treatments such as cognitive behaviour therapy (CBT) and response prevention. Additionally, impaired self-awareness (SA) secondary to TBI can be one of the major barriers to engagement in rehabilitation (Sherer, Bergloff, Levin, High, Oden & Nick, 1998, and may in itself be related to an impairment of executive functions such as error monitoring (Robertson & Schmitter-Edgecombe, 2015), as well as possibly the nature or location of anatomical lesion (s). While some studies have suggested a specific lesion location (Bilgic, Baral-Kulaksizoglu, Hanagasi, Daylan, Aykutlu, Gurvit & Emre, 2004; Berthier, Kulisevsky, Gironell &López, 2000) often frontal, others have implicated the number of lesions (Sherer, Hart, Whyte, Nick, & Yablon, 2005) as being important in SA after TBI. As regards cognitive functions and SA, impaired executive function has been fairly consistently associated with poor SA (Bivona,, Ciurli, Barba, Onder, Azicnuda, Silvestro, Mangano, Rigon, & Formisano, 2008).

In summary, the current study examined the relationship between certain aspects of cognitive functioning, self-awareness, and neuro-imaging findings and OC symptoms and OCPD-like traits in individuals with moderate-severe TBI.

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Specifically, associations between executive functioning (EF), memory, self-awareness, and number of lesions, the presence of frontal lesions, and self-reported OC symptoms and OCPD-like traits were examined.

Method

Participants

Prior to commencement of the study, ethical approval was obtained from the local university and hospital institutions where the study was conducted. Clinicians at a community-based outpatient brain injury rehabilitation service identified potential participants prospectively from their active caseloads. An information sheet was provided (English or Welsh as preferred by the participant). Thirty-eight patients were approached, and 31 agreed to participate in the study, after four declined to take part and three were not contactable. Once written consent was obtained, neuropsychological and questionnaire measures were administered in one session either at the clinic or within participants' homes. Participant travel costs to and from the clinic were reimbursed. Participants were free to withdraw from the project at any time.

Inclusion criteria were a confirmed medical history of moderate-severe TBI, age 18-65 years, duration of 6 months or > since injury, and sufficient cognitive ability to complete neuropsychological and questionnaire measures. Exclusion criteria were previous or current substance misuse requiring treatment, other neurological disorder, pre-morbid psychiatric disorder, or a learning disability, as evidenced from examination of each patient's clinical records. Potential participants were in the first instance approached by their treating clinician to determine if they met the inclusion and exclusion criteria, before providing them with information about the study.

The demographic characteristics of the sample are provided in Table 1. Most participants were males (n = 25) with an average of 12 years of education (M = 12.87, SD = 2.36 years). Neuropsychological assessment was performed in English and all testing administered by the first two authors. All the participants were first-language English speakers.

Injury Information

TBI severity was determined retrospectively from each participant's medical notes. Applying the Mayo Classification System (Malec, Brown, Leibson, Flaada, Mandrekar, Diehl & Perkins, 2007), all participants were deemed to have sustained a definite moderate-severe TBI. Table 2 contains injury severity characteristics and neuroimaging data for each participant. Average time since injury was 9.47 years (SD = 9.14 years; range = 8 months – 40 years). Road traffic collisions accounted for 41.9% of injuries (n=13) with the remainder falls (35.5%, n=11), assaults (12.9%, n=4), blast-related trauma (6.5%, n=2), and industrial accident (3.2%, n=1).

Table 1.
Participant Demographic Characteristics

	M ± SD
Age (years)	44.71 ± 15.90
Education (years)	12.87 ± 2.36
	n (%)
Gender (% males)	25 (80.6)
Employment Status	
Unemployed	14 (45.2)
Employed	12 (38.7)
Student	4 (12.9)
Retired	1 (3.2)
Time Since Injury	
8 months – 3 years	8 (25.8)
3 – 6 years	6 (19.4)
6 – 9 years	3 (9.60)
9 – 13 years	8 (25.8)
13+ years	6 (19.4)

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Table 2.

Clinical Markers of Injury Severity

Participant	GCS Score	LoC	PTA	Scan report	Number of Lesions	Frontal Lesion(s)
1	—	2 days	7days	Left frontal and parietal lobe lesions	2	Yes
2	6	—	35 days	Right-sided acute subdural haematoma	1	No
3	—	2 days	—	Occipital titanium plate inserted	1	No
4	7	—	—	Occipital skull fracture, extradural haematoma	2	No
5	15	—	—	Low-density abnormality on tip of thalamus (left side), small anterior thalamic hypodensity	2	No
6	15	No LoC	—	Right occipital lobe, extra-axial collection, subarachnoid blood	3	No
7	3	—	—	Normal CT	0	No
8	8	—	7 days	Small bilateral acute haematoma, left temporal lobe contusion	3	No
9	9	—	182 days	Subarachnoid haemorrhage, right parietal bone fracture, right extradural haematoma and contusions	4	No
10	11	—	—	Right parietal skull fracture, bilateral acute haemorrhage, coup-contrecoup injury	3	No
11	14	—	—	Temporal skull fracture, right extradural and left intracerebral haematoma	3	No
12	6	—	—	Small subdural bleed in left temporo-parietal region, occipital fracture, small frontal contusions (left side)	4	No
13	9	—	—	Right parietal skull fracture, base of skull fracture (right side), bilateral temporal lobe contusions, shallow left subdural haematoma	5	No
14	9	—	20 days	Small subarachnoid haemorrhage, haemorrhage near the vertex	2	No
15	14	—	7 days	Skull fracture, right frontal contusion and small subdural haematoma in right parietal lobe	3	Yes
16	3	21 days	56 days	Blood over tentorium cerebelli, blood in both posterior horns	2	No
17	3	—	—	Small right frontal and left occipital contusions, right frontal subarachnoid haemorrhage, DAI	4	Yes
18	3	—	21 days	Right frontal lobe and left temporal contusions, haemorrhage in subcortical white matter and subcortical frontal lobe (both left side), DAI	5	Yes
19	—	—	—	Right temporal contusions, subdural haematoma	3	No
20	3	—	—	Subarachnoid haemorrhage, parietal bone fracture (left side), multiple petechial haemorrhages, DAI	4	No
21	14	—	—	Right frontal lobe contusion and bleed	2	No
22	8	—	—	DAI	1	No
23	—	—	42 days	Fractured skull, left fronto-temporal subdural haematoma	2	No
24	9	—	—	DAI	1	No
25	3	—	—	Left frontal extradural haematoma, right fronto-	9	Yes

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				parietal extradural haematoma, left anterior temporal subdural haematoma, bilateral frontal, left occipital and left temporal contusions, traumatic SAH, skull base fracture		
26	—	—	1 day	Significant left frontal contusion, depressed left frontal fracture, small haemorrhage	3	Yes
27	—	—	7 days	Normal CT	0	No
28	—	12	—	No scan report	4	No
29	14	—	—	Haematoma left fronto-temporal and haemorrhage left frontal	2	Yes
30	10	—	—	Large right fronto-temporal extradural haematoma and midline shift	2	Yes
31	—	—	10 days	No scan report	—	—

Note. No data available; GCS = Glasgow Coma Score; LoC = Loss of consciousness (days); PTA = Posttraumatic Amnesia (days); CT = Computed Tomography; DAI = Diffuse Axonal Injury

Measures

Neuropsychological Tests

This study investigated executive functioning and certain aspects of self-directed planning on a visuo-spatial copying task.

Executive Function.

Trail Making Test. Set-shifting was assessed using the Trail Making Test (TMT; Reitan, 1958). The TMT taps a variety of cognitive abilities including visuo-motor tracking and divided attention, and is often used as a measure of set-shifting. Time taken (seconds) to complete trail A and trail B were recorded. We calculated a new measure—TMT-ratio score—which provides an estimate of the relative slowing experienced in TMT-B (i.e., the shifting condition) as a function of baseline speed in TMT-A (i.e., baseline). Higher ratio scores indicate more impaired performance in the shifting condition (i.e. TMT-B) relative to own baseline; ratio scores closer to 1 suggest more equal performance across both conditions (and hence little additional cost of shifting).

Rey Complex Figure. Test The Rey Complex Figure Test (RCFT, Osterrieth, 1944) is a commonly used measure in TBI populations and assesses a range of cognitive abilities including planning and strategy, visuospatial recall, and visuospatial constructional ability. Participants are required to copy (Copy Condition) a complex figure containing 18 separate elements, prior to two recall conditions. In the current study, four performance measures were calculated for the Copy Condition: a) an Accuracy score using scoring criteria of two points per correctly reproduced element; range 0 – 36 Osterrieth, 1944); b) Time taken to copy (seconds); c) Strategy score (Strategy was scored independently by the first and third authors, using the system described by Hamby, Wilkins & Barry, 1993); and d) an Efficiency (IES) score, which assesses speed-accuracy trade-off. The IES was calculated as IES = RT / (1 – PE), where RT is the time taken to complete the figure, and PE is the proportion of errors made. Higher scores reflect more accurate but slower performance.

New learning and retention

We examined auditory learning and retention using the Rey Auditory Verbal Learning Test (RAVLT, Schmidt 1996).

Rey Auditory Verbal Learning Test. The RAVLT provides a measure of verbal memory and learning. The test is suitable for assessing short-term auditory verbal memory, retroactive and proactive interference, as well as yielding information on retention of information. Participants listened to a list of 15 words repeated over five trials and recalled as many words as possible following each trial. Retention was assessed after a delay of approximately 30-minutes (Delayed Recall condition). A recognition condition was also included, where participants were required to correctly identify the 15 target words in amongst 35 distracter words (Recognition condition). Three outcome indices were used: (a) Total Acquisition: sum of words correctly recalled over trials 1-5 (range 0 – 75); (b) Delayed Recall: number of words correctly recalled after 30-minute delay

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(range 0 – 15); and (c) Recognition: number of words correctly identified minus number of incorrectly identified words (range 0 – 15).

Questionnaires Participants completed two questionnaires, each assessing obsessive-compulsive symptoms and OCPD-like traits.

Obsessive-Compulsive Disorder

Florida Obsessive-Compulsive Inventory. Florida Obsessive-Compulsive Inventory (FOCI, Storch, Bagner, Merlo, Shapira, Geffken, Murphy & Goodman, 2007) is a brief self-report questionnaire containing separate scales for obsessive-compulsive symptom enumeration (Symptom Checklist), and evaluation of symptom severity (Symptom Severity). Participants mark the presence or absence of 20 common obsessions and compulsions, and a Symptom Checklist score is subsequently derived by summing the 20 items (range = 0 – 20). Higher scores indicate greater OCD symptomatology. If a participant endorses two or > symptoms, the severity of the endorsed symptom(s) are then rated according to five areas (time occupied, interference, distress, avoidance, and degree of control) on a scale of 0 – 4. A Symptom Severity score is obtained by summing the five severity items (range = 0 – 20), with higher scores indicating greater symptom severity.

Obsessive-Compulsive Personality Disorder

The Schedule for Nonadaptive and Adaptive Personality-2. The Schedule for Nonadaptive and Adaptive Personality-2 26 (SNAP) is a 375-item true/false instrument assessing a dimensional model of personality disorder (PD). In the current study, only items from the OCPD PD (23 items) scale were administered. The OCPD scale assesses characteristics associated with OCPD including perfectionism ('I don't consider a task finished until its perfect'), and cognitive and/or behavioural rigidity (e.g. 'I'm rather set in my ways'). A Total score for the OCPD (range = 0 – 25) scale is obtained by summing the total items the participant endorses. Higher score indicates greater symptomatology.

Self-awareness

Awareness Questionnaire. Participants independently completed the patient version of the Awareness Questionnaire (AQ, Clark, 1993) and their respective treating clinician completed the clinician version. The AQ consists of three forms (patient, clinician, and significant other versions). Self-rated and "significant other" forms have 17 identical items while the clinician form has one additional item (18 in total). The additional item measures clinician perceptions of patient's self-awareness.

On each form, the current abilities of the person with TBI, as compared to before the injury are rated on a five-point scale ranging from "much worse" to "much better". The discrepancy score was used an indication of participants' level of self-awareness. Participants' scores were subtracted from clinicians' scores to produce a positive or negative difference score. Negative values on this measure indicate that a patient under-emphasises their difficulties whilst positive values suggest an over-estimation of functioning, following TBI.

Neuroimaging data

The study looked at neuroimaging data of participants. The narrative from scan reports in participants' medical records were used to determine a) the total number of reported lesions, and b) whether patients had documented frontal involvement secondary to TBI or not.

Procedure

The 31 participants in this study completed neuropsychological tests and questionnaires to assess aspects of executive function, new learning and retention, speed-accuracy ratios on cognitive tasks, self-awareness, and the presence of OCD symptoms and OCPD-like traits. Neuroimaging data were analyzed to capture number of brain lesions reported, and if frontal lesions were present or not (see Table 2).

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Results

Data Preparation

All analysis was conducted using R statistics. The distributions of all dependent variables (see Table 3) were examined visually for adherence to normality. Where the assumption of normality was not met, a transformation was applied to the raw data. The transformations, where relevant, are shown in Table 3. Means and standard deviations for neuropsychological task performance are presented in Table 4. Descriptive statistics obtained on clinical measures are presented in Table 5.

Table 3.

Transformations used when dependent variable was not normally distributed. "x" refers to an individual data point.

Dependent Variable	**Transformation**
FOCI-Symptom	$\text{Log}(x + C^a)$
FOCI-Severity	$\text{Log}(x + C^a)$
SNAP-OCPD	—
TMT-B/A Ratio	Log(x)
RCFT Accuracy Copy	—[b]
RCFT Copy Time	—
RCFT IES	Log(x)
RCFT Strategy Scores	—
AQ	—
RAVLT Total	$-\text{Log}(-x + C_1) + C_2$[c]
RAVLT Delay	—
RAVLT Recognition	$-\text{Log}(-x + C_1) + C_2$[d]
Number of Lesions	$\text{Log}(x + C^a)$
Lesion Location	—

Note. [a]The constant C was defined as 1 – min(variable), where min(variable) refers to the smallest value in the set of scores for the variable. [b]The constant C was defined as max(variable) + 1, where max(variable) refers to the largest value in the set of scores for the variable. [b]There was one extreme outlier in this variable which reduced the normality of the data. We left this variable untransformed as we had no reason to remove this outlier, but examined the influence of this outlier in the analysis section. [c]This transformation requires two constants. C_1 is a constant set to ensure that the lowest value of $(-x + C_1)$ is equal to 1. C_2 is a constant set to ensure that the lowest value of the transformed variable is equal to 1. For these data, $C_1 = 62$ and $C_2 = 4.892$. [d]This transformation is the same as the previous one, but with different values for the constants. For these data, $C_1 = 16$ and $C_2 = 3.708$. FOCI = Florida Obsessive-Compulsive Inventory; SNAP = Schedule for Nonadaptive and Adaptive Personality; TMT B/A Ratio = Trail Making Test B/A Ratio Score; RCFT = Rey-Osterrieth Complex Figure Test; RCFT IES = RCFT Efficiency Score; AQ = Awareness Questionnaire; RAVLT = Rey Auditory Verbal Learning Test.

Table 4.

Means and Standard Deviations for Neuropsychological Measures (non-transformed)

Measure	*M ± SD*	*Range*	*Percentile Average*
Executive Function			
RCFT			
Accuracy	34.29 ± 4.85	9 - 36	
Strategy			
Time (seconds)	167.75 ± 64.59	69 – 314.4	
IES			
TMT			
B/A Ratio	2.34 ± 1.13	1.18 – 7.17	
New Learning & Retention			

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RAVLT		
Total Acquisition	42.61 ± 11.99	13 - 61
Delayed Recall	8.43 ± 3.03	3 - 13
Recognition	10.68 ± 3.50	1- 15

Note. RCFT = Rey-Osterrieth Complex Figure Test; IES = RCFT Efficiency Score; TMT B/A Ratio = Trail Making Test B/A Ratio Score; RAVLT = Rey Auditory Verbal Learning Test

Table 5.

Descriptive Statistics for Clinical and Personality Characteristics

Measure	*M ± SD*	*Range*
Obsessive-Compulsive Disorder		
FOCI		
Symptom	4.48 ± 3.89	0 - 12
Severity	4.52 ± 4.46	0 - 17
Obsessive-Compulsive Personality Disorder		
SNAP	13.48 ± 4.33	6 - 23
Self-Awareness		
AQ		

Note. FOCI = Florida Obsessive-Compulsive Inventory; SNAP = Schedule for Nonadaptive and Adaptive Personality; AQ = Awareness Questionnaire

Analysis Strategy

There are three aspects we expand upon below regarding our analytical approach: a) Controlling for demographic variables; b) Use of Bayesian analysis; and c) Issues surrounding multiple comparisons.

Controlling for demographic variables. First, we wished to establish whether any of the primary dependent variables were associated with any of the demographic variables: Age, Years of Education, and Time Since Injury. An initial correlation was conducted between the primary dependent variables and the demographic data, which indicated the following:

1. Age was correlated with: RCFT Copy Time [r(31) = 0.61, p<.001]; RCFT IES [r(31) = 0.54, p<.01]; and AVLT Delay [r(31) = -0.45, p<.05].
2. Education did not correlate with any primary dependent variable.
3. Time Since Injury correlated with FOCI-Symptom [r(31) = 0.37, p<.05]; TMT-B/A Ratio [r(31) = -0.40, p<.05]; and RCFT Copy Time [r(31) = 0.36, p<.05].

Any analysis reported below with a primary dependent variable that correlated with a demographic variable was conducted using a partial correlation controlling for that demographic variable.

Bayesian analysis. Secondly, due to our relatively low sample size, we were concerned regarding the possibility of uninformative null results (i.e., non-significant correlations). In the traditional null hypothesis significance-testing framework (NHST; i.e., utilising p-values), a non-significant effect leaves the researcher in a "state of suspended disbelief" (Wetzels & Wagenmakers, 2012, p.1059). That is, a non-significant effect could have occurred because there is no true effect to detect, or that the data collected were unable to detect the effect. One cannot arbitrate between these accounts using standard NHST, as it is incorrect to conclude on the basis of a non-significant p-value that the null hypothesis has been supported; that is, with p-values, one cannot provide support for the null hypothesis.

To overcome this issue, we complemented our presentation of standard NHST correlation measures with Bayes factors, which allow the researcher to quantify evidence for or against the null hypothesis. The Bayes factor—indicated as BF10—provides an estimate of the relative plausibility of the observed data under two competing hypotheses. In our example, the

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two hypotheses we are assessing are the presence of a correlation (H1) and the absence of a correlation (H0). The Bayes factor is a ratio score that is directly interpretable in terms of which model is more likely, given the observed data. Bayes factors greater than one indicate more support for H1, and Bayes factors between zero to one indicate support for H0.

Bayes factors thus provide the relative strength of evidence in the data in support of one hypothesis over an alternative hypothesis. A Bayes factor between 1 and 3 (or 1 and 0.33) indicates just anecdotal (i.e., very weak) support for H1 (or H0, respectively). Bayes factors between 3 and 10 (or 0.33 and 0.10) represent moderate support for H1 (or H0); Bayes factors between 10 and 30 (or 0.10 and 0.033) represent strong support for H1 (or H0); and Bayes factors greater than 30 (or less than 0.033) represent very strong support for H1 (or H0). Utilisation of Bayes factors thus allows the researcher to overcome the "state of suspended disbelief" with standard non-significant effects because the strength of support for the null can be established. For all analyses reported below, we use standard NHST correlation and partial correlation methods; we complement these analyses with Bayes factors for correlations and partial correlations using the methods outlined in Wetzels and Wagenmakers (2012).

Multiple comparisons. We did not control for multiple comparisons due to the exploratory nature of our study. This has precedence in the literature along similar lines of investigation as the current work (see for example Berthier, Kulisevsky, Gironell & López, 2000; Rydon-Grange & Coetzer, 2017). However, for completeness, we indicate in Table 6 which significant correlations would survive such a correction as Bonferroni. It is important to note that multiple comparisons are not an issue for Bayesian analysis and as such no corrections are made in Table 7.

Table 6.
Pearson product–moment correlation coefficients (*r*)—or partial correlation coefficients, where indicated—between dependent variables and the FOCI Symptom, FOCI Severity, and SNAP OCPD scales. Total N was 31, except where stated otherwise.

Dependent Variable	**FOCI Symptom**	**FOCI Severity**	**SNAP OCPD**
TMT-B/A Ratio	-0.21	-0.03	-0.15
RCFT Accuracy Copy[a]	-0.27	-0.33	-0.01
RCFT Copy Time[b]	0.05	0.11	0.33^{+}
RCFT IES[b,c]	0.26	0.31^{+}	0.21
RCFT Strategy[d]	**-0.59****[e]	**-0.51****	0.17
AQ	-0.33^{+}	-0.31^{+}	-0.35^{+}
RAVLT Total	0.07	-0.09	-0.20
RAVLT Delay[b]	0.14	-0.05	-0.25
RAVLT Recognition	0.34^{+}	0.28	-0.23
No. Lesions[f]	-0.27	-0.27	-0.17
Lesion Location[g]	0.27	0.11	0.08

Note: $^{+}$p<0.1; *p<0.05; **p<0.01. [a]These data had an extreme outlier with an accuracy score of 9 (all others were >30). Removal of this subject did not change the qualitative pattern of outcome. [b]Controlling for age as a partial correlate. [c]IES = Efficiency score combining speed & accuracy on the RCFT. [d]Based on RC's scores. Note that the agreement between raters (RC and CR) was found to be poor (Cohen's Kappa = 0.15, z = 1.79, p = 0.074). For this anlaysis, N = 29. [e]This correlation remains significant when controlling for multiple comparisons (Bonferroni correction, $\alpha = 0.05/30 = 0.001$). [f] N = 30. FOCI = Florida Obsessive-Compulsive Inventory; SNAP = Schedule for Nonadaptive and Adaptive Personality; TMT B/A Ratio = Trail Making Test B/A Ratio Score; RCFT = Rey-Osterrieth Complex Figure Test; RCFT IES = RCFT Efficiency Score; AQ = Awareness Questionnaire; RAVLT = Rey Auditory Verbal Learning Test. [g]Lesion location data was unavailable for one participant. Therefore, for this analysis, N = 30.

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Table 7.

Bayes factors (BF_{10}) for the correlations—or partial correlations, where indicated— between dependent variables and the FOCI Symptom, FOCI Severity, and SNAP OCPD scales. Total N was 31, except where stated otherwise.

Dependent Variable	FOCI Symptom	FOCI Severity	SNAP OCPD
TMT-B/A Ratio	0.257	0.140	0.188
RCFT Accuracy Copy[a]	0.399	0.697	0.139
RCFT Copy Time[b]	0.186	0.211	0.837
RCFT IES[bc]	0.492	0.754	0.338
RCFT Strategy[d]	**38.989**	**8.143**	0.208
AQ	0.741	0.556	0.883
RAVLT Total	0.148	0.156	0.254
RAVLT Delay[b]	0.253	0.202	0.491
RAVLT Recognition	0.775	0.457	0.290
No. Lesions[e]	0.423	0.385	0.208
Lesion Location[f]	0.400	0.169	0.153

Note: **Bold numbers** indicate moderate-to-strong support for the alternative hypothesis (i.e., presence of a correlation); underlined numbers indicate moderate support for the null hypothesis (i.e., no correlation).[a]These data had an extreme outlier with an accuracy score of 9 (all others were >30). Removal of this subject did not change the qualitative pattern of outcome. [b]Controlling for age as a partial correlate. [c]IES = Efficiency score combining speed & accuracy on the RCFT. [d]Based on RC's scores. Note that the agreement between raters (RC and CR) was found to be poor (Cohen's Kappa = 0.15, z = 1.79, p = 0.074). For this analysis, N = 29. [e]N = 30. FOCI = Florida Obsessive-Compulsive Inventory; SNAP = Schedule for Nonadaptive and Adaptive Personality; TMT B/A Ratio = Trail Making Test B/A Ratio Score; RCFT = Rey-Osterrieth Complex Figure Test; RCFT IES = RCFT Efficiency Score; AQ = Awareness Questionnaire; RAVLT = Rey Auditory Verbal Learning Test. [f]Lesion location data was unavailable for one participant. Therefore, for this analysis, N = 30.

Overview of Results

The Pearson product-moment correlation coefficients (and partial correlations where appropriate) are shown in Table 6. Correlations that reached typical levels of significance are indicated in bold. The Bayes factors (BF10) for the correlations (or partial correlations) are shown in Table 7. In this table, Bayes factors which show at least moderate evidence in support for the presence of a correlation are shown in bold, and Bayes factors which show at least moderate evidence in support of the absence of a correlation are shown as underlined. Note that all other correlations are not informative from a Bayesian perspective (i.e., no clear support for one hypothesis over the other).

TMT-B/A ratio

For this measure, there were small, non-significant negative correlations with FOCI symptom, FOCI severity, and SNAP OCPD scores. Interestingly, the Bayes factors showed that these data provide moderate support for the null hypothesis of no correlation, suggesting these data are informative despite the small sample.

RCFT accuracy copy

For this measure, there were small, non-significant negative correlations with FOCI symptom, FOCI severity, and SNAP OCPD scores. Although the Bayes factors showed more support for the null hypothesis for all three correlations, this evidence was anecdotal (weak) for FOCI symptom and FOCI severity, but was moderate for SNAP OCPD, suggesting support for no correlation for this measure.

RCFT copy time

This measure showed small positive correlations (all non-significant) with FOCI symptom, FOCI severity, and SNAP OCPD scores, although the correlation with SNAP OCPD "approached" significance ($p<0.1$). The Bayes factors showed moderate support for the absence of a correlation for FOCI symptom and FOCI severity. Despite the "approaching" significance of the correlation between RCFT copy time and SNAP OCPD, the Bayes factor actually showed anecdotal (weak) support for the null hypothesis of no correlation.

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RCFT IES

This measure showed small positive correlations (all non-significant) with FOCI symptom, FOCI severity, and SNAP OCPD scores. The Bayes factors for all correlations showed anecdotal (weak) support for the null hypothesis. These analyses are thus inconclusive.

RCFT strategy

This measure showed large negative correlations with FOCI symptom and FOCI severity (both significant), and a small positive correlation with SNAP OCPD. The Bayes factor for showed strong support for the presence of a correlation between RCFT strategy and FOCI symptom, and moderate support for the presence of a correlation between RCFT strategy and FOCI severity. The Bayes factor also showed moderate support for the absence of a correlation between RCFT strategy and SNAP OCPD.

AQ diff

This measure showed small negative correlations (all non-significant) with FOCI symptom, FOCI severity, and SNAP OCPD scores, although all "approached" significance (all $p<0.1$). Despite this, the Bayesian evidence actually showed some support for the null hypothesis of no correlation, although this evidence is anecdotal (weak), and thus these analyses are inconclusive.

AVLT total

This measure showed a small positive correlation with FOCI symptom, and small negative correlations with FOCI severity and SNAP OCPD. The Bayes factors suggest that these data are informative as all provide moderate support for the null hypothesis of no correlation.

AVLT delay

This measure showed a small positive correlation with FOCI symptom, and small negative correlations with FOCI severity and SNAP OCPD. The Bayes factors suggest that the data for FOCI symptom and severity are informative as both provide moderate support for the null hypothesis of no correlation, but the Bayes factor for the correlation between AVLT delay and SNAP OCPD provides anecdotal (weak) support for the null hypothesis.

AVLT recognition

There were small positive correlations between this measure and FOCI symptom and FOCI severity (both non-significant), and a small negative correlation with SNAP OCPD. Only the Bayes factor for SNAP OCPD was informative, providing moderate support for the null hypothesis of no correlation. The Bayesian analysis of the correlations with FOCI measures were anecdotal (weak) support for the null.

Number of lesions

This measure showed small negative correlations (all non-significant) with FOCI symptom, FOCI severity, and SNAP OCPD scores. Only the Bayes factor for SNAP OCPD was informative, providing moderate support for the null hypothesis of no correlation. The Bayesian analysis of the correlations with FOCI measures were anecdotal (weak) support for the null.

Lesion location

Lesion location was coded as a binary variable (1 = frontal involvement; 0 = no frontal involvement). There were small, non-significant, positive correlations between this measure and FOCI symptom, FOCI severity, and SNAP OCPD. The Bayes factor for the null correlations were informative for FOCI severity and SNAP OCPD, providing moderate support for the null hypothesis of no correlation. The Bayes factor for the correlation between lesion location and FOCI symptoms indicate anecdotal (weak) support for the null.

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Discussion

Although anxiety and OCD-type psychiatric presentations after TBI are thought to be associated with poor outcome, our understanding of factors contributing to their presentation in this clinical population remains limited (Mallya, Sutherland, Pongracic, Mainland & Ornstein, 2015). Cognitive impairment is considered to be an important factor underpinning some of the symptoms of OCD (Benzina, Mallet, Burguiere, N'Diaye & Pelissolo, 2016). Interestingly, neuropsychological test performance is associated with poor outcome in those patients with OCD who have not suffered a TBI (Perna, Cavedini, Harvey, Di Chiaro, Dacco & Caldirola, 2016). Previous research by Penades, Catalan, Andres, Salamero and Gasto (2005) suggested that memory impairment in OCD possibly reflects problems with organisational strategies rather than poor recall per se. The main finding from our study suggested strong support for the presence of a correlation between Rey Complex Figure (RCFT) strategy and the Florida Obsessive-Compulsive Inventory (FOCI) symptom scores, and moderate support for the presence of a correlation between RCFT strategy and FOCI severity scores.

This finding from our study can potentially be interpreted as providing partial evidence that executive impairment may underpin some of the problem's patients with OCD experience. The interpretation should however be cautious. When considering the speed component of executive functions, tests of executive function relying on self-initiated strategies to organise a task of copying a complex geometrical design is completed slower by patients with OCD (Roth, Baribeau, Milovan & OConnor, 2004). Our study did not support a problem with speed being present in our patient group. Furthermore, cognitive flexibility is considered to be an important function to examine in OCD, including set shifting, reversal and alternation, cognitive control, trials to shift and motor inhibition among others (Gruner, & Pittenger, 2017). OCPD is associated with poor cognitive flexibility and impaired planning (Fineberg, Day, de Koenigswarter, Reghunandanan, Kolli, Jefferies-Sewell, Hranov, & Laws, 2015). Whereas Paast, Khosravi, Memari, Shayestehfar, & Arabi, 2016) found that both OCD and OCPD were impaired on tests measuring cognitive flexibility and planning. Our study failed to provide data supporting the notion that impairment of cognitive flexibility is central to OCD symptoms. The data from our study also suggested an absence of correlation between OCD phenomena and memory impairment, and also between neuroimaging findings and OCD symptoms in this group of TBI patients.

The findings from our study may have some implication for the treatment and rehabilitation of patients who present with OCD-type symptoms after TBI. The treatment of OCD is widely considered to be the SSRIs and CBT (Stein, Ipser, Baldwin & Bandelow, 2007). Where patients present with OCD-type phenomena after TBI, it may be helpful to consider performing neuropsychological testing to determine if any cognitive impairment could be contributing to their presentation. Where cognitive impairment mirroring OCD phenomena is identified in this clinical population, including executive impairment, it may be helpful to offer adjunctive tailored cognitive rehabilitation strategies to try and reduce some of the difficulties stemming from obsessions and compulsions deemed to be mediated in part by their cognitive impairment.

There were several limitations to this study, including small sample size and the wide range of time since injury observed in the group of participants. Questionnaires also only provide self-reported data, rather than an objective clinician assessment of neuropsychiatric difficulties. Furthermore, using discrepancy scores on the AQ to measure self-awareness can be problematic, as most measures of SA can be influenced by other factors, for example mood (Richardson, McKay & Ponsford, 2015). Nevertheless our study did have a few strengths. This is perhaps the biggest group of participants with TBI formally assessed for OCD-type symptomatology and compared with objective neuropsychological test findings. Finally, utilising a Bayesian anlysis of our data, the analysis can not only suggest where there is a correlation present, but also suggest where there may be small meaningful (i.e., informative) null correlations, as well as identify where there are no correlations present. This facilitates the process of learning more from our data and is likely to be important in informing areas for further investigation in future studies investigating OCD spectrum presentations after TBI.

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